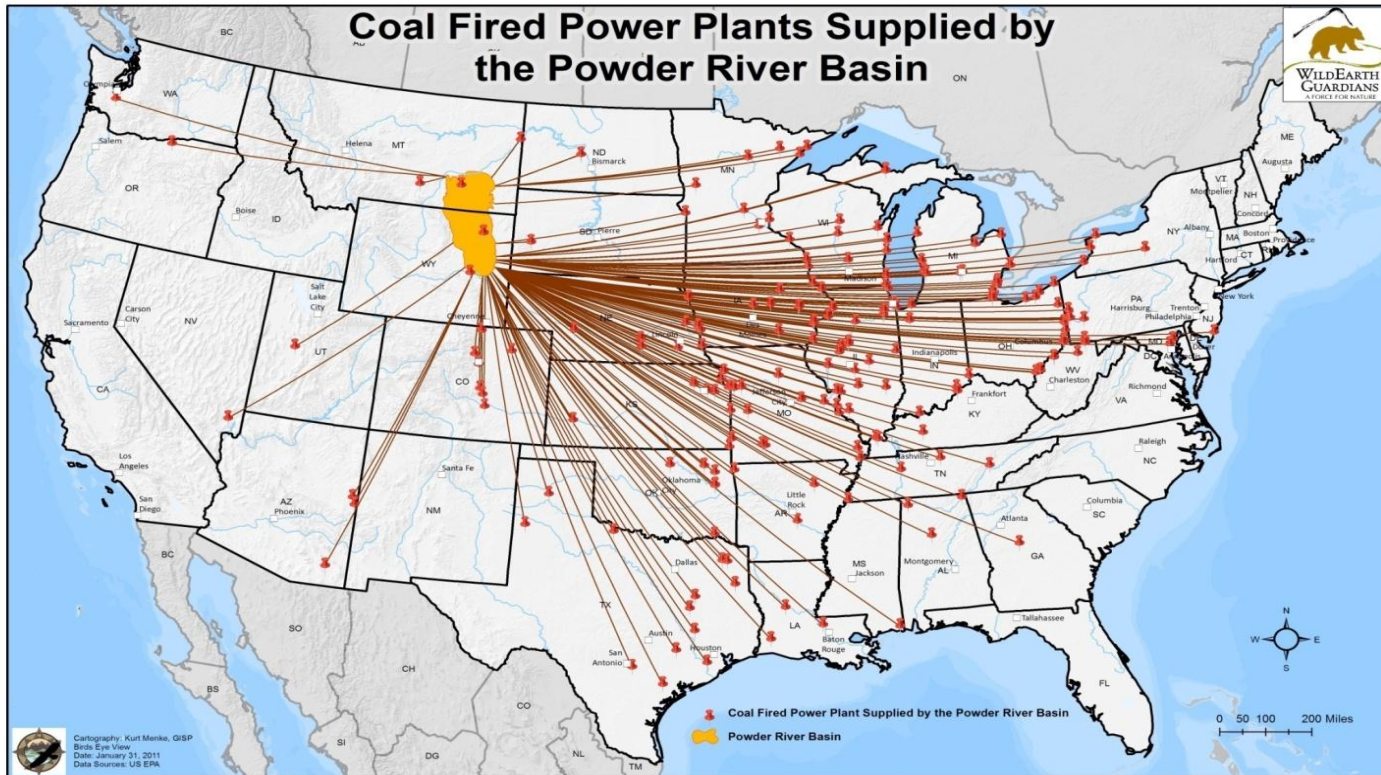


Public Health Impacts of Coal Exports

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Pulmonary Specialist
Billings Clinic

May 10th, 2013

Powder River Basin Coal



In 2009, 42% of the nation's coal came from the Powder River Basin

99% of this coal was used in the US

Source: Energy Information Administration

U.S. Coal Markets Declining

Coal consumption has declined >20% since 2000

No new power
plants built

Transition to
natural gas

Renewable/
sustainable
energy use



Colstrip Power Plant, MT

Growing Demand in Asia

Growth in Asian coal demand is expected to dramatically increase by 2030.

US Energy Information Agency predicts that nearly 90% of that increased use will be in China.



Beijing, China

Predicted Increases in Coal Export

Recent PRB coal export shipments: 3 and 6 million tons/year

Based on coal company projections, PRB coal exports are expected to increase to:

- 75 million tons/year by 2017
- 170 million tons/year by 2022



75 million tons of coal = 30 additional trains/day going through Montana



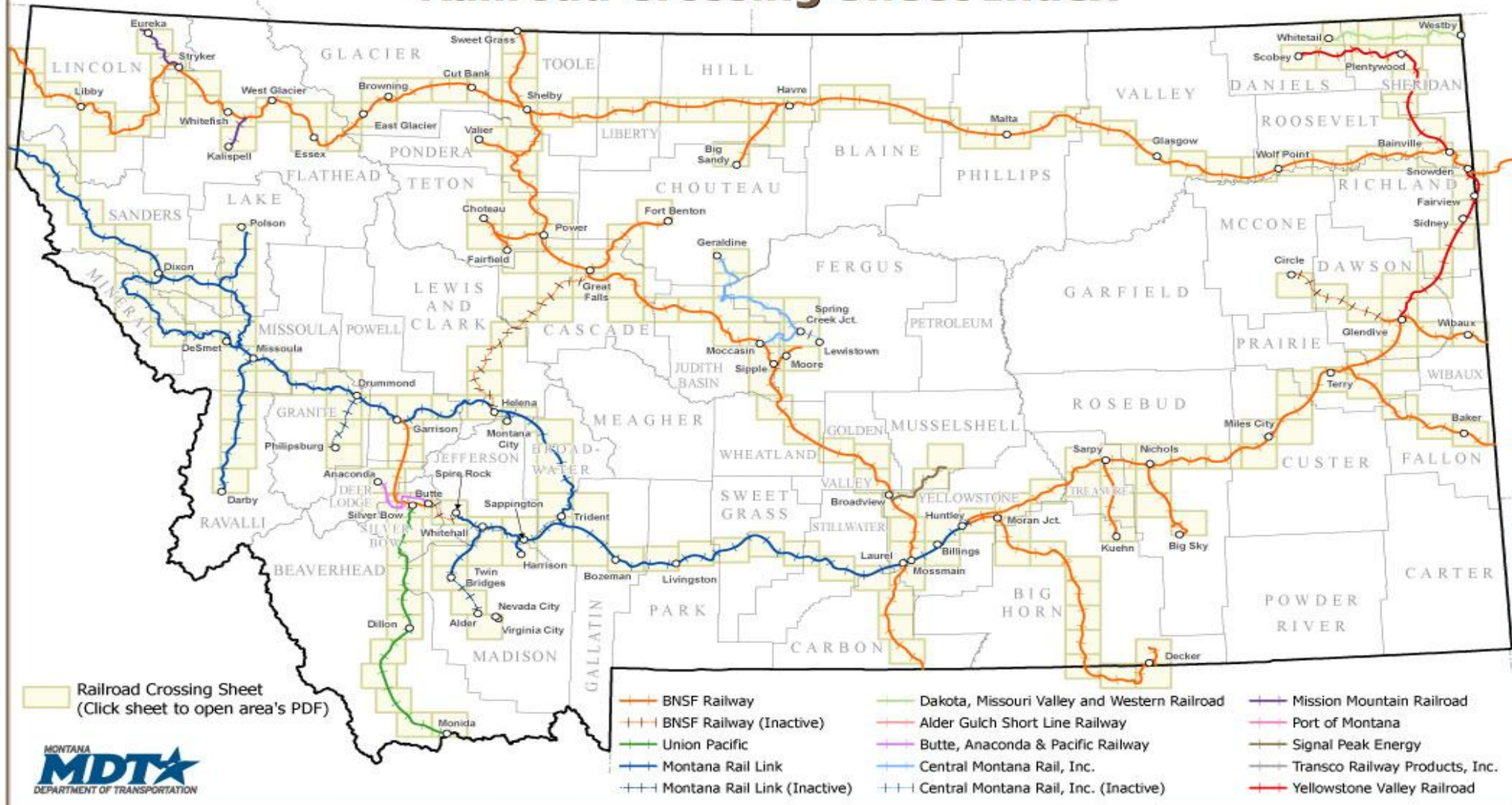
15 full trains
going West

- 120 – 125 cars/train
- 115 tons of coal/car
- 14,000 tons of coal/train

15 empty trains
going East

170 million tons of coal = 64 additional trains/day

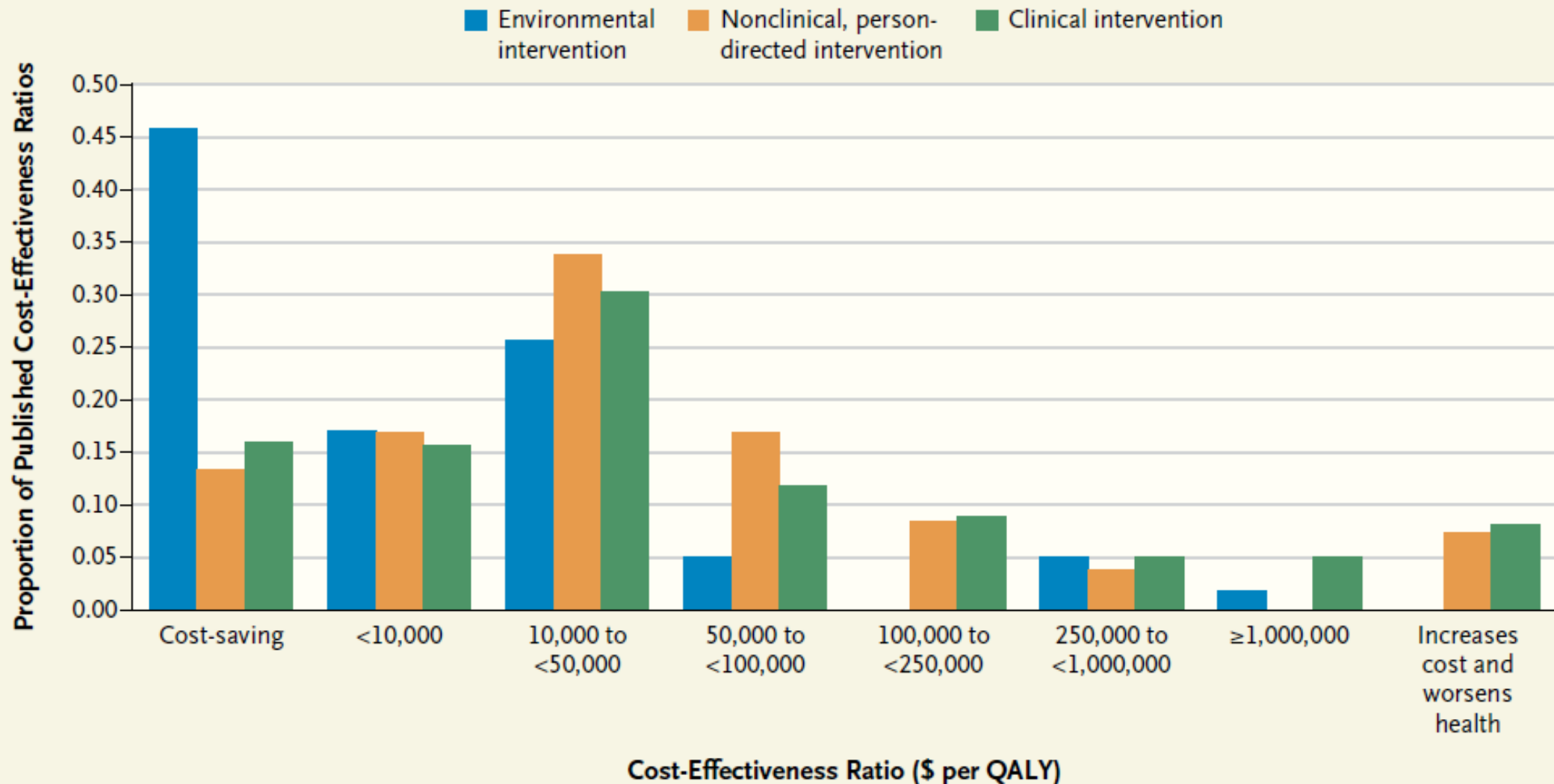
Railroad Crossing Sheet Index



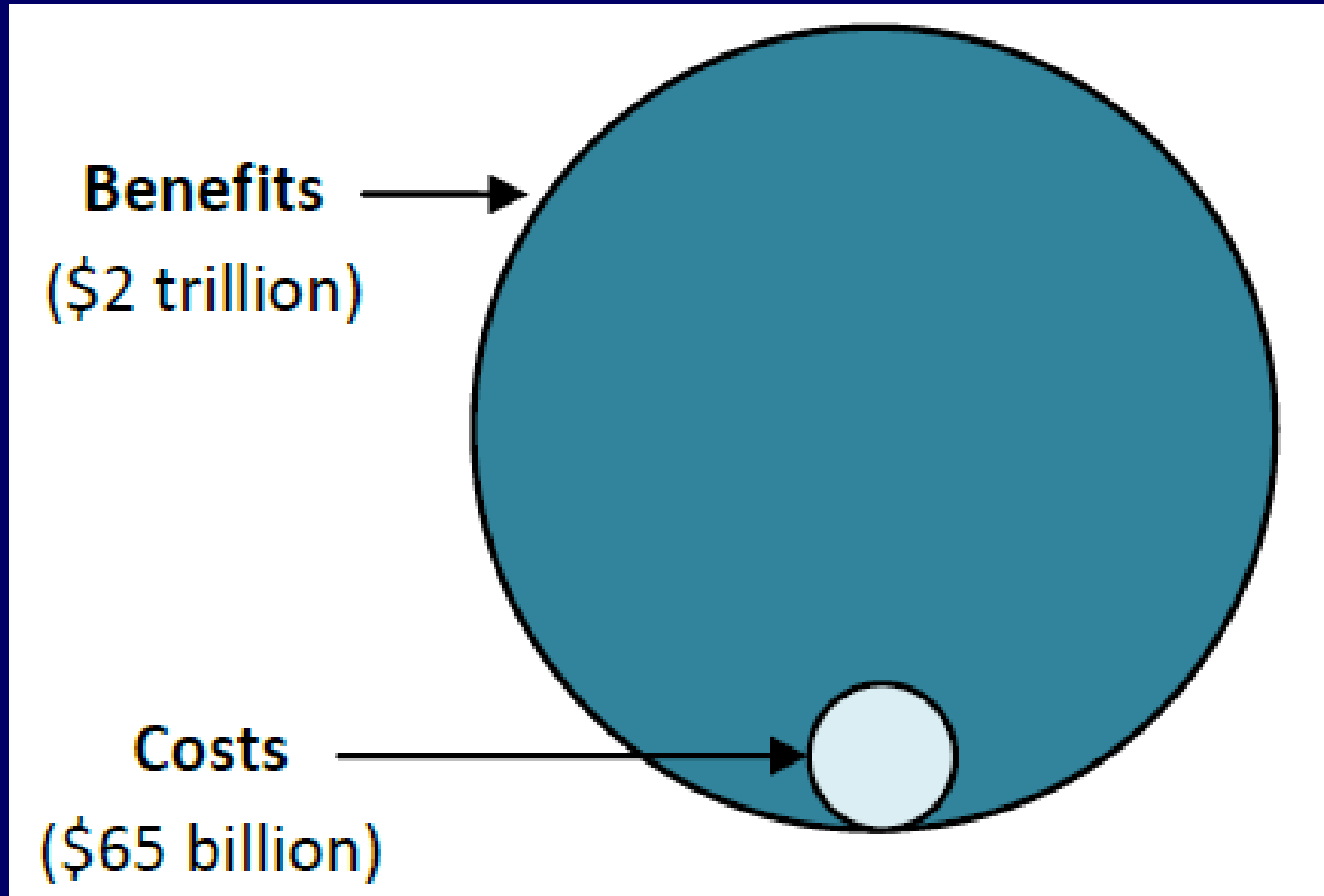
Why worry?

- People's health will be adversely affected
 - Asthma
 - COPD
 - CVAs and MIs
 - HTN
 - Lung Cancer
- Economics
 - Health effects represent a “negative externality” to the coal industry
 - Disease prevention through environmental intervention is cost saving

The Cost-Effectiveness of Environmental Approaches to Disease Prevention



Clean Air Act Amendments of 1990



Sources of Impact from Coal Trains

- Diesel Exhaust
- Coal dust
- Noise
- Global Climate Change

Sources of Impact

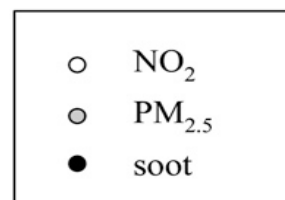
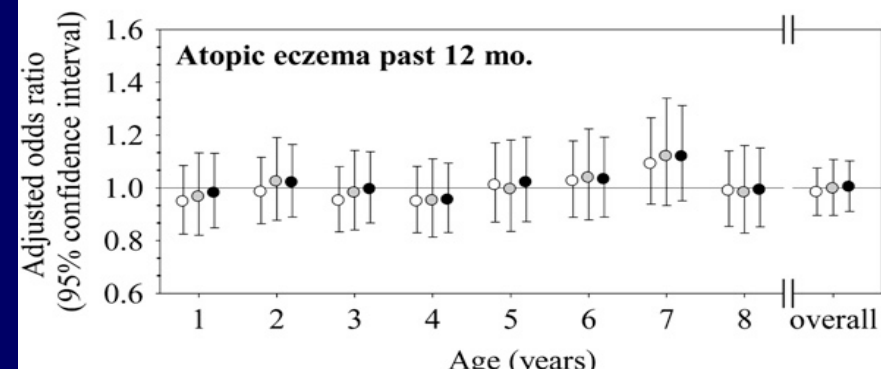
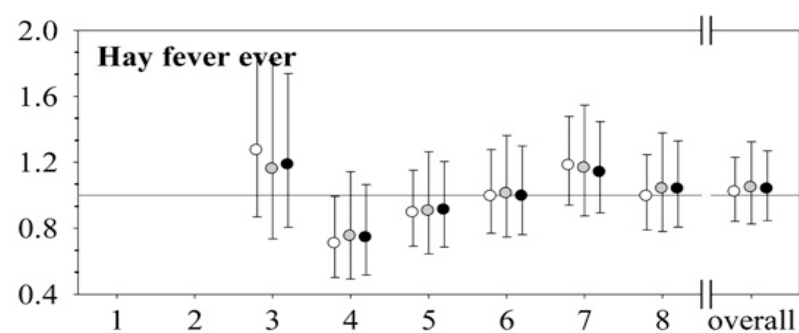
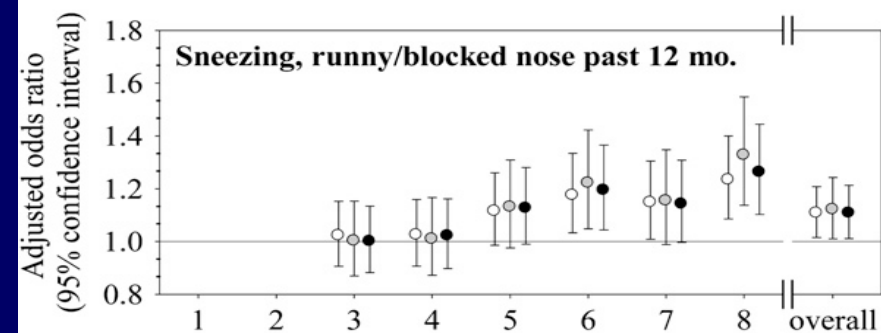
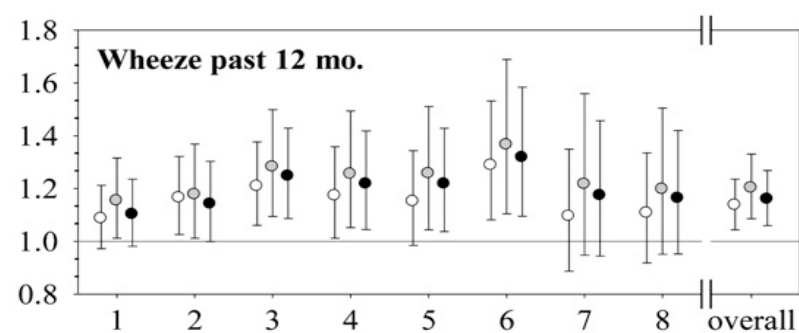
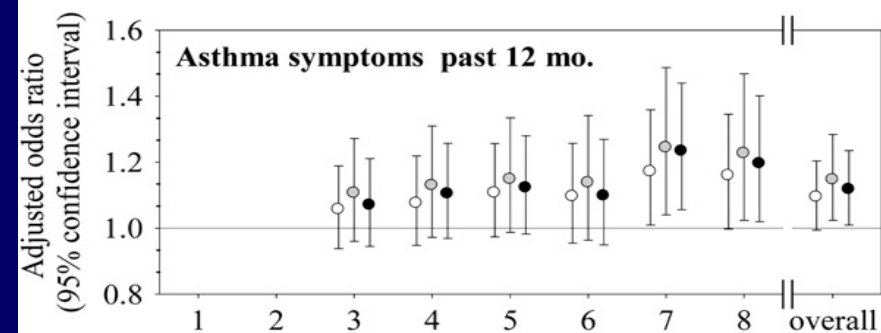
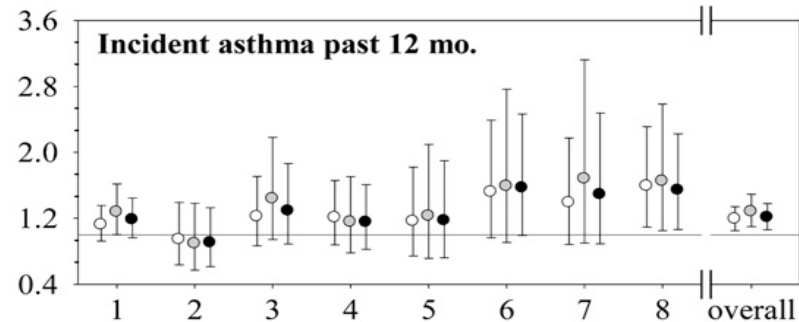
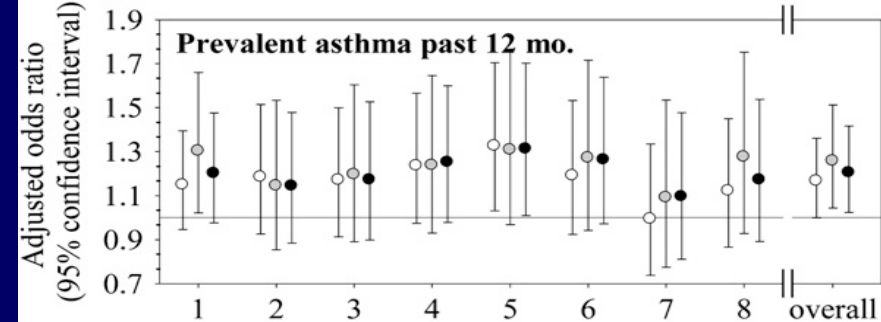
- Diesel Exhaust
 - PM 2.5
 - Nitrogen Oxides (NO_x)
 - Ozone
 - Benzene
 - Very fine particles absorb organics onto their surface and carry these deep into the lung
- Coal Dust
 - PM 2.5
 - Carbon
- Noise
 - dB
- Climate change
 - Temperature, growing season, precipitation, CO₂ levels

Health Effects

- Causes Asthma
- Exacerbations of Asthma and COPD
- Vascular Disease
 - MI
 - CVA
 - Cognitive decline
- Hypertension
- Cancer

Traffic-related Air Pollution and the Development of Asthma and Allergies during the First 8 Years of Life

- Prospective birth cohort study with 8 yr follow-up of 3863 children.
- Determined individual exposures to
 - Particulate matter (PM_{2.5})
 - Nitrogen Dioxide (NO₂)
 - Soot
 - Land use regression model
- Yearly questionnaires Sxs, Diagnoses.
- Compared odds ratio of interquartile range increase in pollutants



Gehring,
 AJRCCM.
 2010; 181:
 596

Traffic-related Air Pollution and the Development of Asthma and Allergies during the First 8 Years of Life

TABLE 5. ADJUSTED ASSOCIATIONS* BETWEEN PM_{2.5} LEVELS AT BIRTH ADDRESS AND HEALTH OUTCOMES FOR CHILDREN WHO DID AND CHILDREN WHO DID NOT MOVE HOUSE DURING THEIR FIRST 8 YEARS OF LIFE

	Nonmovers			Movers		
	n [†]	OR	95% CI	n [†]	OR	95% CI
During the first 8 years of life						
Prevalent asthma	1,575 [‡]	1.34	1.03–1.75	1,605 [‡]	1.19	0.92–1.53
Incident asthma	1,551 [‡]	1.36	1.09–1.69	1,588 [‡]	1.20	0.98–1.48
Asthma symptoms	1,556 [‡]	1.27	1.07–1.49	1,596 [‡]	1.05	0.90–1.23
Wheeze	1,575 [‡]	1.25	1.08–1.45	1,605 [‡]	1.15	1.00–1.32
Sneezing, runny/blocked nose	1,556 [‡]	1.14	0.98–1.34	1,596 [‡]	1.09	0.95–1.25
Hay fever	1,556 [‡]	1.43	1.01–2.04	1,596 [‡]	0.81	0.58–1.12
Atopic eczema	1,575 [‡]	1.03	0.89–1.21	1,605 [‡]	0.97	0.84–1.12
At age 8 years						
Bronchial hyperresponsiveness	464	0.99	0.69–1.41	351	0.96	0.69–1.34
Allergic sensitization	617	1.25	0.96–1.61	881	1.07	0.83–1.39

Traffic-related Air Pollution and the Development of Asthma and Allergies during the First 8 Years of Life

- OR of 1.28 for developing asthma for interquartile range increase in traffic related air pollution

Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits

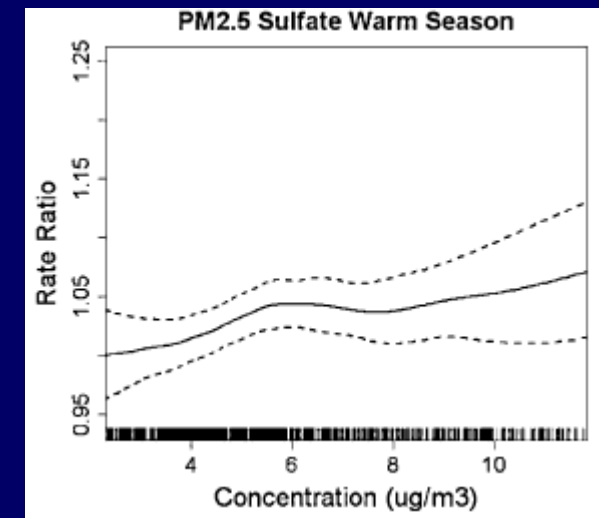
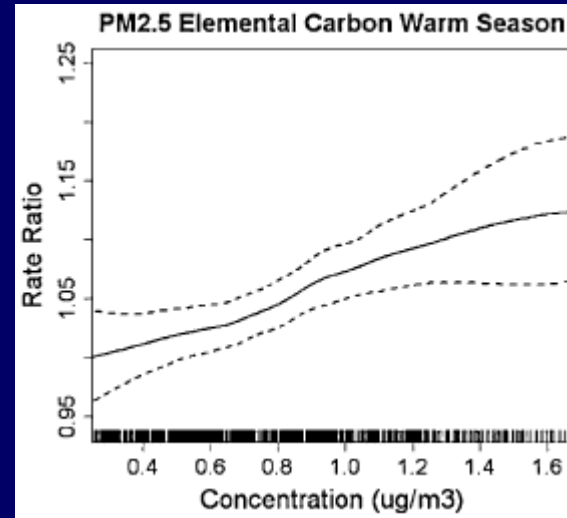
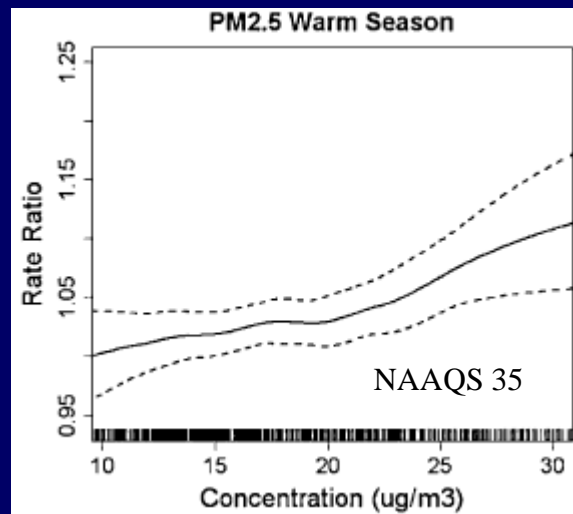
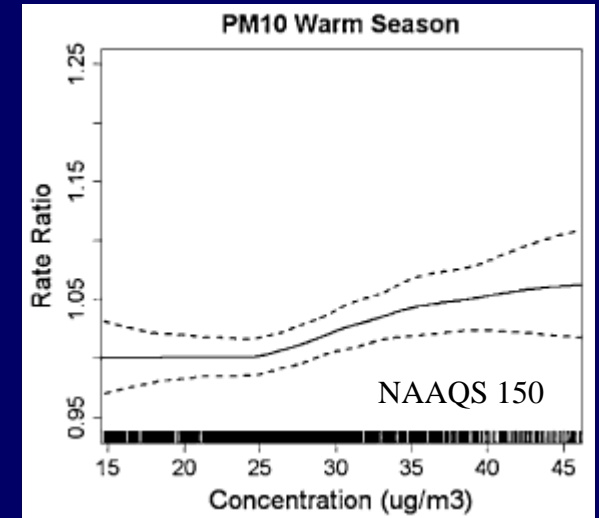
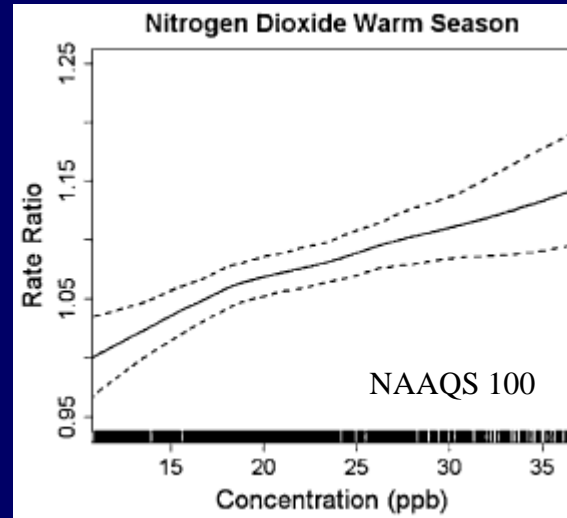
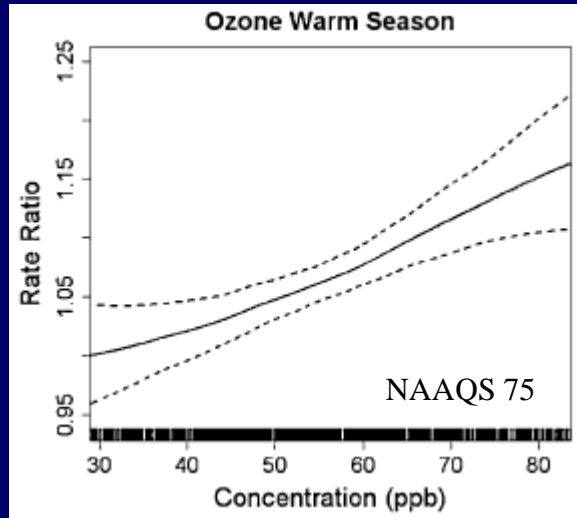
- Compared pediatric ER visits for asthma or wheezing with ambient levels of ozone and traffic related pollutants
 - 41 Atlanta ERs with 91,386 visits
 - 3 day moving average of pollutant level preceding ER
 - 1993-2004
- Case crossover analysis using Poisson generalized linear models

Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits

TABLE 2. RATE RATIOS AND 95% CONFIDENCE INTERVALS FROM POISSON GENERALIZED LINEAR MODELS FOR INTERQUARTILE RANGE INCREASES IN THREE-DAY MOVING AVERAGE POPULATION-WEIGHTED AMBIENT AIR POLLUTANT CONCENTRATIONS*

	Overall RR (95% CI) (Jan–Dec)	Warm Season RR (95% CI) (May–Oct)	Cold Season RR (95% CI) (Nov–Apr)
Ozone ^{††}	1.062 (1.031–1.093)	1.082 (1.043–1.123)	1.044 (0.992–1.098)
Nitrogen dioxide [†]	1.036 (1.018–1.055)	1.066 (1.038–1.095)	1.016 (0.992–1.040)
Carbon monoxide [†]	1.023 (1.006–1.041)	1.068 (1.034–1.102)	1.005 (0.985–1.025)
Sulfur dioxide [†]	1.012 (0.994–1.030)	1.030 (1.002–1.058)	1.001 (0.978–1.025)
PM ₁₀ [§]	1.020 (1.003–1.038)	1.026 (1.001–1.051)	1.018 (0.994–1.043)
PM _{10–2.5}	1.034 (1.011–1.057)	1.025 (0.991–1.059)	1.041 (1.010–1.073)
PM _{2.5}	1.020 (1.002–1.039)	1.043 (1.016–1.070)	1.005 (0.978–1.031)
PM _{2.5} sulfate	1.014 (0.995–1.033)	1.027 (1.004–1.049)	0.991 (0.953–1.029)
PM _{2.5} elemental carbon	1.015 (0.997–1.033)	1.041 (1.010–1.072)	1.003 (0.981–1.026)
PM _{2.5} organic carbon	1.008 (0.994–1.021)	1.034 (1.007–1.062)	1.000 (0.985–1.016)
PM _{2.5} water-soluble metals	1.021 (1.000–1.042)	1.029 (1.003–1.055)	1.005 (0.968–1.043)

Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits



Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits

- Traffic related pollutants increase ER visits for asthma in children.
 - Occurred at exposures significantly below current standards

Recent warming by latitude associated with increased length of ragweed pollen season in central North America

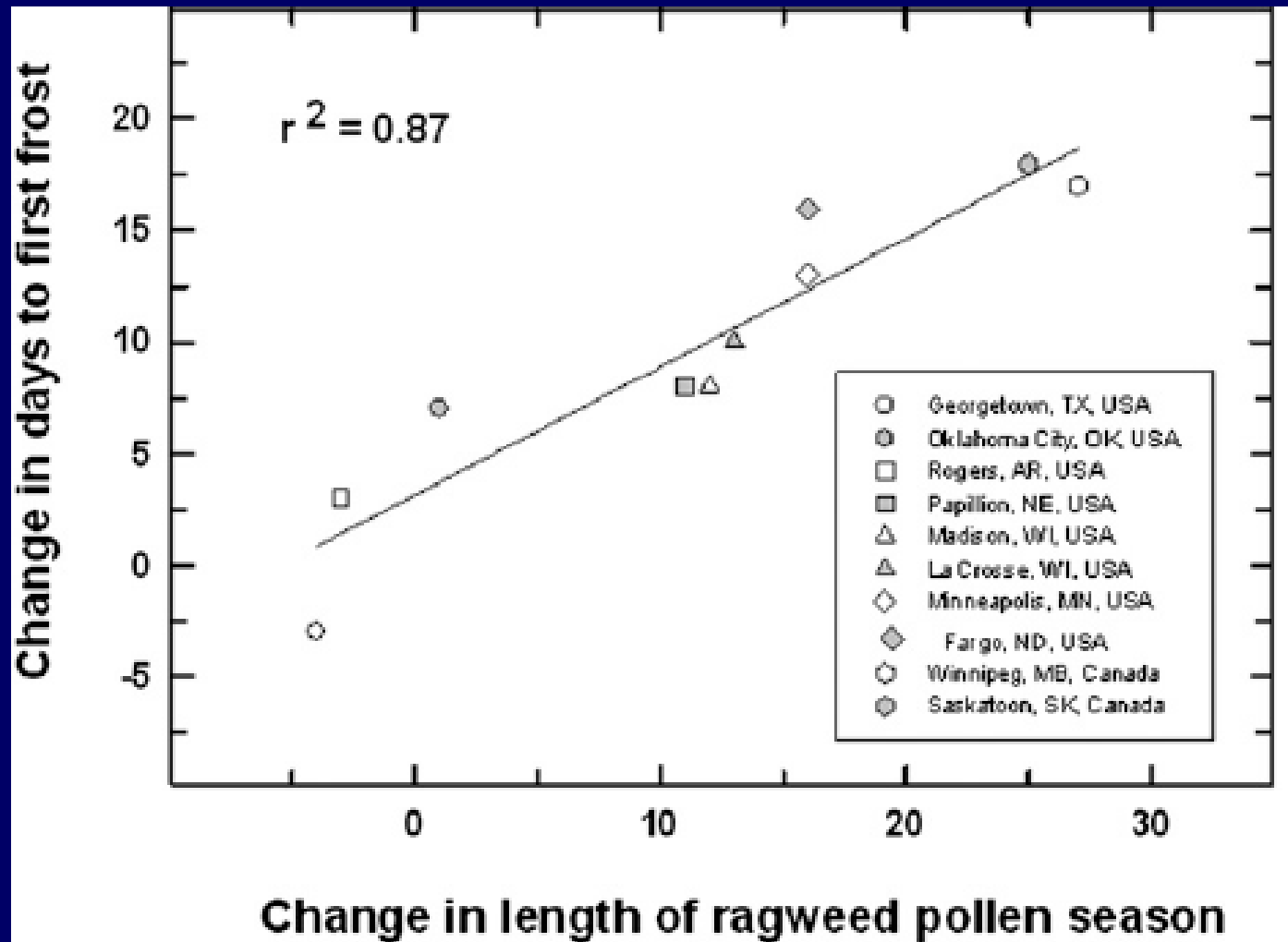
Table 1. Change in length (day of year, days) of ragweed pollen season as a function of latitude for National Allergy Bureau and Aerobiology Research Laboratories sites along a south–north latitudinal gradient

Location	Latitude	Years of data	Start	End	Start	End	Change
			1995		2009		
Georgetown, TX	30.63°N	17	198 ± 7	320 ± 7	195 ± 7	313 ± 7	–4 d
Oklahoma City, OK	35.47°N	19	212 ± 7	300 ± 10	227 ± 9	316 ± 15	+1 d
Rogers, AR	36.33°N	15	231 ± 7	295 ± 8	227 ± 6	296 ± 8	–3 d
Papillion, NE	41.15°N	21	212 ± 3	281 ± 6	208 ± 4	288 ± 10	+11 d
Madison, WI	43.00°N	27	208 ± 2	272 ± 4	205 ± 3	281 ± 6	+12 d
LaCrosse, WI	43.80°N	22	213 ± 3	271 ± 3	205 ± 5	276 ± 5	+13 d*
Minneapolis, MN	45.00°N	19	208 ± 5	270 ± 6	206 ± 7	284 ± 7	+16 d*
Fargo, ND	46.88°N	15	216 ± 4	252 ± 8	217 ± 4	269 ± 8	+16 d*
Winnipeg, MB, Canada	50.07°N	16	207 ± 7	264 ± 6	197 ± 7	279 ± 7	+25 d*
Saskatoon, SK, Canada	52.07°N	16	206 ± 12	250 ± 6	197 ± 13	268 ± 7	+27 d*

Years represent the number of years for which pollen data were available. Regression analysis was used to determine the “best-fit” line for all years for a given location. This analysis was then used to determine the start and end day of each year ($\pm 95\%$ confidence interval) for the duration of the ragweed pollen season in 1995 and again in 2009.

*Significant increase in the length (days) of the ragweed pollen season.

Recent warming by latitude associated with increased length of ragweed pollen season in central North America



Ambient Air Pollution and the Risk of Acute Ischemic Stroke

- Evaluated the association between PM 2.5 and ischemic stroke
 - Time stratified case-crossover design
 - 1763 consecutive neurologist confirmed ischemic strokes admitted to BIDMC 1999 to 2008
 - During this time Boston was “in attainment”
 - Hourly levels of PM 2.5 in the time preceding CVA
 - Excluded days when the air quality was poor (but still in attainment)

Ambient Air Pollution and the Risk of Acute Ischemic Stroke

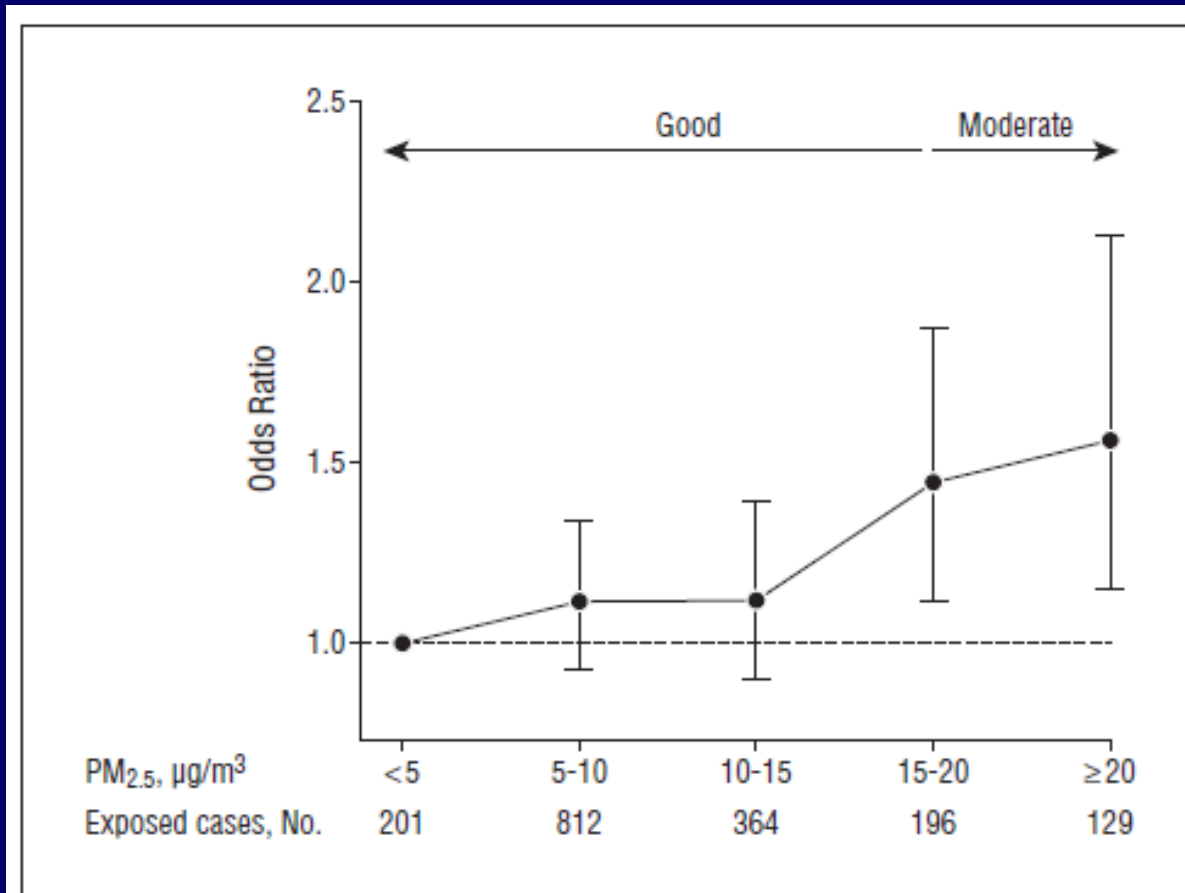


Figure 1. Odds ratio of ischemic stroke onset for US Environmental Protection Agency categories (*good* and *moderate*) of mean ambient fine particulate matter air pollution (PM_{2.5}) levels in the 24 hours preceding stroke onset. Error bars indicate 95% CIs.

Ambient Air Pollution and the Risk of Acute Ischemic Stroke

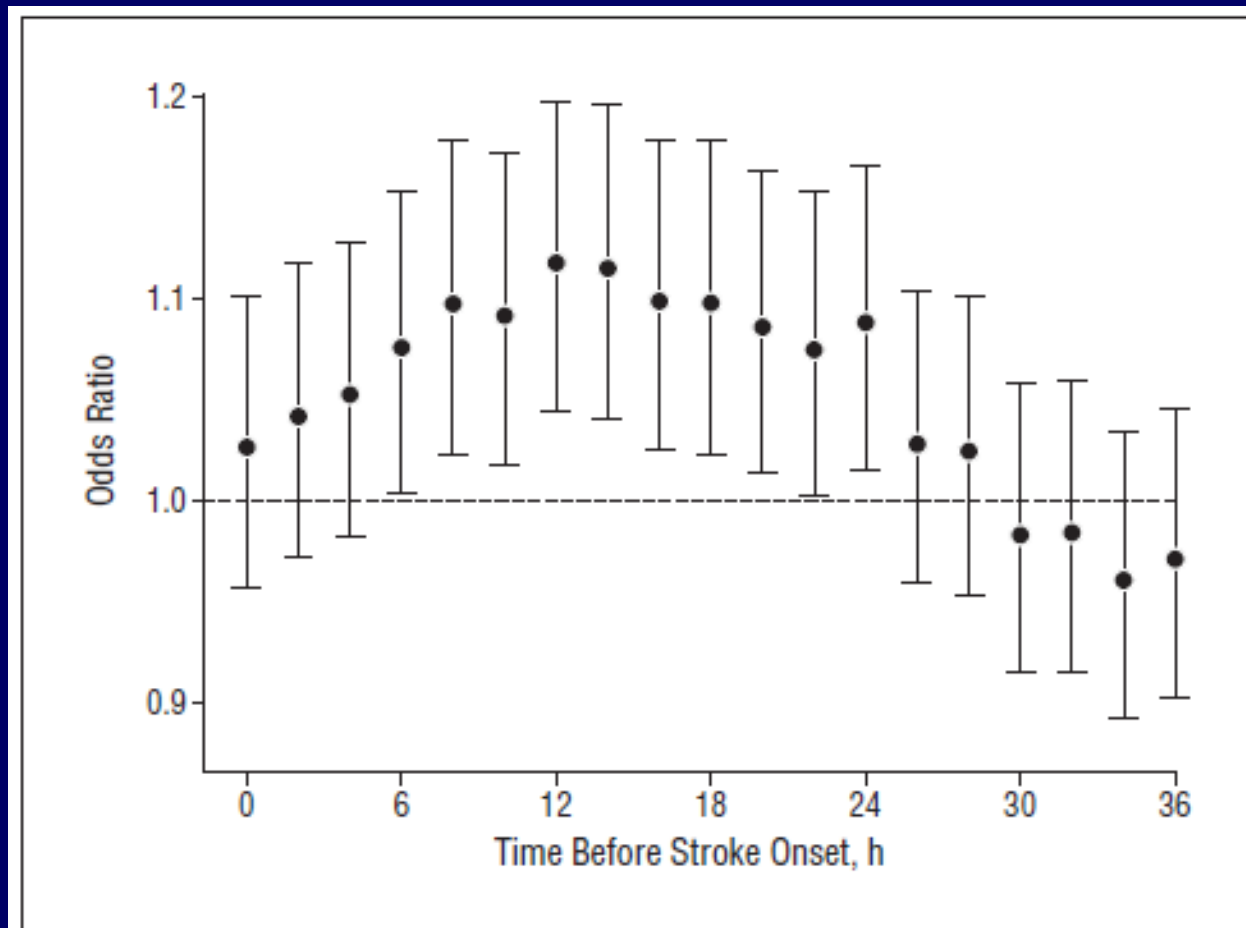


Figure 2. Odds ratio of ischemic stroke onset per interquartile range increase in concentration of ambient fine particulate matter air pollution ($6.4 \mu\text{g}/\text{m}^3$) in the hours preceding stroke onset. Error bars indicate 95% CIs.

Ambient Air Pollution and the Risk of Acute Ischemic Stroke

- Ischemic strokes are strongly associated with increased PM 2.5 exposures well within the national standards
 - 34% increase in risk of stroke for moderate vs mild exposures peaking 12 hours after the exposure.

Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: a cohort study

- Prior studies linking traffic and railway noise did not control for air pollutants.
- 57,053 enrolled
 - Baseline: BP and health survey
 - 5-7 yr follow-up: health survey
 - Modeled traffic and railway noise and NO_x exposure
 - Day, evening and night

Exposure to road traffic and railway noise and associations with blood pressure and selfreported hypertension: a cohort study

- Traffic noise – 0.26 mmHg increase in SBP
- Railway noise > 60 dB – 8% increase in developing HTN

The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust

- Nested case control study in a cohort of 12315 non-metal miners, 8 facilities
 - 198 Lung Cancer deaths
 - 562 Matched controls
- Exposures to “respirable elemental carbon” modeled from detailed occupational history
- Adjusted for smoking and other potential confounders

The Diesel Exhaust in Miners Study

Table 3. Odds ratios (ORs) and 95% confidence intervals (CIs) for average and cumulative REC and total duration REC exposure*

Exposure metric	Case subjects	Control subjects	OR (95% CI)	<i>P</i> _{trend}
Average REC intensity, quartiles, unlagged, µg/m³				
0 to <1	49†	166	1.0 (referent)	.025
1 to <32	50	207	1.03 (0.50 to 2.09)	
32 to <98	49	145	1.88 (0.76 to 4.66)	
≥98	50	148	2.40 (0.89 to 6.47)	
Quartiles, lagged 15 y, µg/m³				
0 to <1	47†	190	1.0 (referent)	.062
1 to <6	52	187	1.11 (0.59 to 2.07)	
6 to <57	49	141	1.90 (0.90 to 3.99)	
≥57	50	148	2.28 (1.07 to 4.87)	
Cumulative REC, quartiles, unlagged, µg/m³-y				
0 to <19	49	151	1.0 (referent)	.083
19 to <246	50	214	0.87 (0.48 to 1.59)	
246 to <964	49	147	1.50 (0.67 to 3.36)	
≥964	50	154	1.75 (0.77 to 3.97)	
Quartiles, lagged 15 y, µg/m³-y				
0 to <3	49	158	1.0 (referent)	.001
3 to <72	50	228	0.74 (0.40 to 1.38)	
72 to <536	49	157	1.54 (0.74 to 3.20)	
≥536	50	123	2.83 (1.28 to 6.26)	
Duration of REC exposure, y				
Unexposed‡	48	165	1.0 (referent)	.043
0 to <5	51	169	1.16 (0.53 to 2.55)	
5 to <10	20	95	0.88 (0.38 to 2.03)	
10 to <15	31	107	0.93 (0.39 to 2.21)	
≥15	48	130	2.09 (0.89 to 4.90)	

The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust

Smoking intensity (packs per day)	Cumulative REC lagged 15 years OR (95% CI), No. of case subjects/No. of control subjects		
	Tertile 1, 0 to < 8 µg/m³-y	Tertile 2, 8 to < 304 µg/m³-y	Tertile 3, ≥304 µg/m³-y
Never smoker	1.0 (referent), 3/59	1.47 (0.29 to 7.50), 4/74	7.30 (1.46 to 36.57), 7/45
<1	6.25 (1.42 to 27.60), 10/41	7.42 (1.62 to 34.00), 10/49	16.35 (3.45 to 77.63), 15/39
1 to <2	10.16 (2.55 to 40.53), 29/78	11.58 (2.87 to 46.71), 32/86	20.42 (4.52 to 92.36), 27/63
≥2	26.79 (6.15 to 116.63), 19/22	22.17 (4.84 to 101.65), 15/22	17.38 (3.48 to 86.73), 10/28
Unknown†	4.13 (0.74 to 23.22), 4/25	3.79 (0.64 to 22.41), 4/23	27.85 (5.03 to 154.31), 9/12

The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust

- 2.83 fold increase in lung cancer from diesel exposures (highest vs lowest quartile)
- 7.30 fold increase in lung cancer in the non-smokers
- Harm was seen at levels analogous to those found in many cities

Variability

- type of train
- Proximity
- Time
- Weather
- individual susceptibility
- Age of train (2008 EPA standards)

Conclusions

- Increased train traffic will increase
 - Asthma problems,
 - MI CVA
 - Cancer
 - By how much is unknown
- The problems will be greatest for those living or working near the railways
- The problems will lessen over the next 20 years due to 2008 EPA locomotive standards.
- But will not go away

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